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Risk factors for male breast cancer (United States)

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***Objectives*: The etiology of male breast cancer is obscure, although an excess risk has been associated with Klinefelter syndrome, testicular disorders, benign breast disease including gynecomastia, use of exogenous estrogens, radiation, and a family history of male or female breast cancer. We conducted a case-control study to investigate risk factors further for breast cancer in men.**

***Methods*: Based on data from the 1986 National (United States) Mortality Followback Survey (NMFS) of almost 20,000 deceased adults (age 25 years or over), we compared information obtained from next-of-kin interviews of 178 men who died of breast cancer with that of 512 male controls who died of other causes. Information was obtained on selected demographic and other factors, including diet, exercise, occupation, height and weight, and use of tobacco and alcohol.**

***Results*: Increased risks were found for men who were described by their next-of-kin as very overweight (odds ratio [OR] = 2.3, 95 percent confidence interval [CI] = 1.1-5.0). The risks associated with the three upper quartiles of body mass index (BMI) (wt/ht^2) were 1.3, 1.6, and 2.3, respectively, with a significant dose-response relationship ($P < 0.01$). An excess risk was also associated with limited exercise (OR = 1.3, CI = 0.8-2.0). Consumption of red meat was associated with an increased risk, and consumption of fruits and vegetables with a decreased risk, although the trends were not significant. No association was found for tobacco or alcohol use, but an excess risk was associated with higher levels of socioeconomic status (SES) (OR = 1.8, CI = 1.1-3.0).**

***Conclusions*: Our study suggests that obesity increases the risk of male breast cancer, possibly through hormonal mechanisms, while dietary factors, physical activity, and SES indicators also deserve further investigation.** *Cancer Causes and Control* 1998, 9, 269-275

Key words: Alcohol, diet, male breast cancer, obesity, tobacco, United States.

Introduction

Male breast cancer is a rare tumor, accounting for 0.8 percent of all breast cancer in the United States and 0.2 percent of all male cancer.[1] It is estimated that 1,400 new cases and 290 deaths in 1997 will be attributed to breast cancer in men.[1] The worldwide variation of male breast cancer resembles that of breast cancer in women, with high rates in North America and Europe and low rates in Asia.[2] The histologic types of male and female breast cancer are also similar, with infiltrating ductal carcinoma being the most common.[3] The five-year relative survival rates for male breast cancer (ranging from 38 to 73 percent) are lower than the corresponding rates for women, since breast cancer in men usually is detected at a later stage.[3]

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The etiology of male breast cancer is obscure.[4] A familial tendency has been reported, occasionally in association with female breast cancer and with mutations of the BRCA2 gene.[5,6] In addition, about six percent of men born with Klinefelter syndrome (XXY) eventually develop breast cancer, with a 20-fold increased risk.[7,8] Further evidence for a role of hyperestrogenism comes from associations with various testicular disorders, gynecomastia, and liver cirrhosis.[9-18] Some reports have suggested that occupational exposures to high temperature and electromagnetic fields (EMF) may increase the risk of male breast cancer,[19-22] but the findings are not consistent.[23,24]

In this case-control study of male breast cancer, we evaluated the role of selected demographic, lifestyle, and anthropometric factors, using data from the 1986 US National Mortality Followback Survey (NMFS).

Materials and methods

Study subjects were selected from 18,733 decedents included in the 1986 NMFS, conducted by the US National Center for Health Statistics (NCHS). Details of this survey have been reported elsewhere.[25-27] Briefly, a 10 percent systematic sample of 1986 US death certificates, excluding Oregon because of consent requirements, was sent by each of the states to NCHS. From these death certificates, a probability sample comprising approximately one percent of adult deaths (aged 25 years and over) was selected. In addition, for several rare cancers (male breast, nasopharynx, small intestine, nasal cavity, adrenal gland, and liver among young women), all deaths in 1985 among Whites and Blacks aged 25 to 74 years were included in the survey.

Questionnaires were sent to the next-of-kin of the decedents to obtain selected information on demographic factors, occupation, height and weight, dietary patterns, physical activity, and use of cigarettes and alcohol. Dietary patterns were assessed by frequency of usual intake of five food groups during the subject's adult life, including red meat (beef, pork, lamb, or hamburger), eggs or dairy products (milk, cheese, or butter), fruits, vegetables, and salt-cured/smoked foods (bacon, hot dogs, or smoked fish). Information was obtained on duration and amount of use of cigarettes, snuff, and chewing tobacco, and on the frequency and amount of alcohol consumption (beer, wine, or liquor). The response rate for the informant questionnaire was 89 percent.[25]

A total of 201 deaths from male breast cancer (ICD-9 code 175)[28] were identified from the 1986 NMFS study (20 from 1986 and 181 from 1985). After excluding 23 subjects who were either Hispanic, or older than 75 years of age, or whose next-of-kin were nonrespondents, 178 cases were available for this analysis.

Controls were selected from male decedents dying of causes other than breast cancer and whose next-of-kin responded to the informant questionnaire. Excluded were 4,854 subjects who died of smoking- or alcohol-related causes, 1,556 subjects who died of any of the five rare cancers with the NMFS study, and 49 non-White subjects other than Blacks. Based on the age distribution of the index cases, up to four controls per case were randomly selected from among the 1,332 eligible con-

Table 1. Odds ratios (OR) and 95% confidence intervals (CI) for male breast cancer by selected characteristics; United States

Selected characteristics	Cases (n = 178)	Controls (n = 512)	OR	(CI)
Type of respondent				
Spouse	96	286		
Parent	28	61		
Child	7	33		
Sibling	24	52		
Other	23	75		
Age at death (yrs)				
25-49	28	107	1.0	—
50-59	43	113	1.4	(0.8-2.5)
60-69	71	169	1.6	(1.0-2.6)
70-74	36	123	1.1	(0.6-1.9)
Race				
Black	17	68	1.0	—
White	161	444	1.4	(0.8-2.5)
Marital status (at time of death)				
Never married	19	66	1.0	—
Divorced/separated	20	75	0.9	(0.5-1.9)
Widowed	17	39	1.5	(0.7-3.2)
Married	118	324	1.3	(0.7-2.2)
Education level (years)				
< 9	31	107	1.0	—
9-11	32	98	1.1	(0.6-2.0)
12	54	151	1.2	(0.7-2.0)
>12	51	132	1.3	(0.8-2.2)
Annual family income in 1985				
< $9,000	38	128	1.0	—
$9,000-$21,999	44	141	1.0	(0.6-1.7)
$22,000+	57	122	1.6	(1.0-2.5)
Unknown	39	121	1.1	(0.6-1.8)
Assets at death				
< $5,000	39	134	1.0	—
$5,000- $49,999	36	136	0.9	(0.5-1.5)
$50,000+	69	146	1.6	(1.0-2.6)
SES index[a]				
Low	32	122	1.0	—
Medium	78	243	1.2	(0.8-1.9)
High	63	131	1.8	(1.1-3.0)

[a] Socioeconomic status = combination of education, annual family income, and assets at death.

trols matched on race and age (five-year age groups). In total, 512 controls were included in the analysis. The highest proportion of deaths among controls was due to malignancies (35 percent), including hematopoietic system ($n = 41$), brain ($n = 31$), prostate ($n = 62$), rectum ($n = 14$), melanoma ($n = 14$), and other types of cancers ($n = 16$), followed by external causes of injury and poisonings (11 percent), diabetes (eight percent), infectious and parasitic diseases (eight percent), nervous system disorders (seven percent), and other conditions (31 percent).

Odds ratios (OR) and corresponding 95 percent confidence intervals (CI) for male breast cancer in relation to potential risk factors were estimated using multiple logistic regression analysis.[29] Risks were assessed for all respondents combined and separately for spouse-respondents only. Potential confounding effects of age, education, annual family income, socioeconomic status (SES), and marital status were examined and adjusted for if necessary. The SES index combined information about education, family income, and assets at death. The body mass index (BMI) was derived by dividing weight (kilograms) by the square of height (meters).

Results

The median age at death was 62 years for the 178 cases and the 512 controls. As shown in Table 1, cases and controls were similar by type of respondent, race, and marital status. However, cases had higher levels of education, annual family income, and assets at death. No excess risk of male breast cancer was observed for cigarette smokers (OR = 0.9, CI 0.6-1.3) or alcohol users (OR = 0.9, CI 0.6-1.6) (Table 2). There was no risk gradient associated with intensity or duration of smoking or drinking.

Table 3 presents risk in relation to height, usual adult weight, BMI, perceived obesity, and level of exercise by type of respondent. Taller men had a slightly higher risk, with no significant dose-response relationship. Men perceived as very overweight by their next-of-kin had a twofold risk (CI = 1.1-5.0). Compared with men in the lowest quartile of usual adult weight, those in the second, third, and highest quartiles had ORs of 1.4, 1.7, and 2.7, respectively, with a significant dose-response relationship ($P < 0.01$). Relative to the lowest quartile of BMI, those in the second, third, and fourth quartiles had elevated risks of 30 percent, 60 percent, and 130 percent, respectively. In addition, men with hardly any exercise had a 1.3-fold risk (CI = 0.8-2.0) compared with those who exercised regularly. A 2.9-fold risk was attained for those in the highest quartile of BMI and with little or no exercise (CI = 1.3-6.6). Risk patterns were similar regardless of type of respondent, although when limiting the analysis to spousal respondents, the risks associated with BMI were more pronounced.

Table 4 shows the risks associated with selected dietary factors. Those who consumed red meat seven or more times a week had a 1.8-fold risk (CI = 0.6-4.9), although the trend was not significant. Intake of fruits and vegetables was associated with a nonsignificantly reduced risk. There were no clear risks related to use of dairy products or salt-cured/smoked foods.

Table 2. Odds ratios (OR) and 95% confidence intervals (CI) for male breast cancer in relation to smoking and drinking; United States

	Cases[a] ($n = 178$)	Controls[a] ($n = 512$)	OR[b]	(CI)
Tobacco				
Never	41	108	1.0	—
Ever	130	387	0.9	(0.6-1.3)
Smokeless only	5	9	1.5	(0.5-4.7)
Ever cigarette smokers	125	378	0.9	(0.6-1.3)
Non-regular	4	25	0.4	(0.1-1.3)
Regular smokers				
< 15 cigarettes/day	25	92	0.7	(0.4-1.3)
15-34 cigarettes/day	64	171	1.0	(0.6-1.5)
35+ cigarettes/day	24	71	0.9	(0.5-1.6)
Alcohol (no. of drinks/day)				
Nonusers	22	62	1.0	—
Ever user	151	434	0.9	(0.6-1.6)
1	41	119	0.8	(0.5-1.6)
2	39	97	1.1	(0.6-2.0)
3-4	28	85	0.9	(0.5-1.8)
5+	31	95	0.9	(0.5-1.8)

[a] Missing data are not included.
[b] Adjusted for age at death and socioeconomic status.

Table 3. Odds ratios (OR) and 95% confidence intervals (CI) for male breast cancer in relation to height, weight, body mass index (BMI), and level of exercise, by type of respondent. United States

	All respondents				Spouse respondents			
	Cases[a]	Controls[a]	OR[b]	(CI)	Cases[a]	Controls[a]	OR[a,b]	(CI)
Total	178	512			95	277		
Height (inches)								
Q1[c] (59-67)[d]	35	126	1.0	—	20	69	1.0	—
Q2 (68-70)	53	166	1.1	(0.7-1.8)	26	89	0.9	(0.4-1.8)
Q3 (71-72)	49	118	1.4	(0.8-2.3)	31	73	1.3	(0.6-2.5)
Q4 (73-82)	33	77	1.5	(0.8-2.6)	18	46	1.1	(0.5-2.4)
Perceived body weight (by next-of-kin)								
About right	92	289	1.0*	—	46	150	1.0	—
Underweight	6	40	0.5	(0.2-1.2)	2	21	0.3	(0.1-1.4)
Somewhat overweight	58	145	1.2	(0.8-1.8)	39	97	1.3	(0.8-2.1)
Very overweight	13	19	2.3	(1.1-5.0)	7	10	2.6	(0.9-7.6)
Usual adult weight (lbs)								
Q1[c] (98-155)[d]	25	26	1.0**	—	11	61	1.0*	—
Q2 (156-170)	35	115	1.4	(0.8-2.6)	17	65	1.3	(0.6-3.1)
Q3 (171-190)	50	137	1.7	(1.0-2.9)	33	84	2.0	(0.9-4.2)
Q4 (191-350)	57	102	2.7	(1.5-4.6)	34	67	2.4	(1.1-5.3)
BMI (wt/ht^2)								
Q1[c] (13.3-22.9)[d]	26	118	1.0**	—	8	57	1.0*	—
Q2 (23.0-25.0)	37	120	1.3	(0.7-2.4)	25	70	2.6	(1.1-6.4)
Q3 (25.1-27.3)	45	120	1.6	(0.9-2.7)	29	71	2.7	(1.1-6.5)
Q4 (27.4-129.5)	59	116	2.3	(1.3-3.9)	33	76	3.2	(1.3-7.6)
Exercise								
Regular	36	111	1.0	—	20	72	1.0	—
Irregular	15	78	0.6	(0.3-1.1)	10	51	0.7	(0.3-1.6)
Hardly any	118	300	1.3	(0.8-2.0)	66	158	1.6	(0.9-2.9)
BMI / exercise								
Q1 / regular	9	44	1.0	—	3	24	1.0	—
Q1 / hardly any	16	73	1.1	(0.4-2.8)	5	33	1.2	(0.3-5.8)
Q2 / regular	16	52	1.4	(0.6-3.6)	11	34	2.6	(0.6-10.5)
Q2 / hardly any	21	65	1.6	(0.6-3.8)	14	35	3.4	(0.8-13.5)
Q3 / regular	13	51	1.1	(0.4-2.9)	9	33	1.9	(0.4- 8.0)
Q3 / hardly any	31	68	2.2	(0.9-5.1)	20	38	4.1	(1.1-15.9)
Q4 / regular	12	33	1.7	(0.6-4.7)	7	26	2.1	(0.5-9.2)
Q4 / hardly any	46	81	2.9	(1.3-6.6)	26	49	4.6	(1.2-17.2)

[a] Missing data are not included.
[b] Adjusted for age at death and socioeconmic status.
[c] Quartiles.
[d] Cutoffs for quartiles.
* Linear trend test, $P < 0.05$.
** Linear trend test, $P < 0.01$.

Discussion

Our case-control study revealed that obesity is a significant risk factor for male breast cancer, whether evaluated by usual adult weight, BMI, or perceived overweight. In a previous study of male breast cancer, obesity and rapid weight gain in the third or fourth decades were linked to increased risk.[13] These findings have not been consistently seen for male breast cancer[9] but resemble the results for breast cancer in postmenopausal women. We did not have data on body weight at various periods of life, so the influence of weight change could not be evaluated in our study.

The effects of obesity on male and female breast cancer risk may be mediated by endogenous estrogens, although only limited data are available in men.[30] Among obese men, it has been shown that estrogen production, metabolism, and bioavailability are enhanced.[12] In particular, the levels of circulating estrogens are increased by the aromatization of androgens with conversion of testosterone to estradiol and androstenedione to estrone in peripheral adipose tissue.[12] Adi-

Table 4. Odds ratios (OR) and 95% confidence intervals (CI) for male breast cancer in relation to food intake, by type of respondent; United States

Foods	All respondents				Spouse respondents			
	Cases[a]	Controls[b]	OR[b]	(CI)	Cases[a]	Controls[b]	OR[b]	(CI)
Total	178	512			95	277		
Red meat (times/wk)								
< 1	5	23	1.0	—	2	11	1.0	—
1-2	21	84	1.2	(0.4-3.4)	11	47	1.5	(0.3-7.9)
3-6	73	193	1.8	(0.6-4.8)	42	129	1.8	(0.4-8.8)
7+	67	180	1.8	(0.6-4.9)	39	94	2.4	(0.5-11.7)
Fruits (times/wk)								
< 1	22	52	1.0	—	12	22	1.0	—
1-2	27	96	0.7	(0.3-1.3)	18	54	0.6	(0.2-1.6)
3-6	45	113	0.9	(0.5-1.6)	25	70	0.6	(0.3-1.5)
7+	73	214	0.8	(0.4-1.3)	40	135	0.5	(0.2-1.2)
Vegetables (times/wk)								
< 1	5	8	1.0	—	1	2	1.0	—
1-2	10	34	0.4	(0.1-1.7)	2	19	0.1	(0.01-1.9)
3-6	23	91	0.4	(0.1-1.2)	12	56	0.2	(0.01-2.5)
7+	129	346	0.5	(0.2-1.7)	80	205	0.3	(0.03-4.2)
Egg/dairy products (times/wk)								
< 1	4	15	1.0	—	2	6	1.0	—
1-2	29	54	2.0	(0.6-6.5)	16	33	1.6	(0.3-9.1)
3-6	36	130	1.0	(0.3-3.3)	23	80	0.9	(0.2-5.1)
7+	97	281	1.3	(0.4-4.2)	54	162	1.1	(0.2-6.0)
Salt-cured/smoked foods (times/mo)								
< 1	61	158	1.0	—	42	102	1.0	—
1-2	59	164	0.9	(0.6-1.4)	35	101	0.8	(0.4-1.3)
3-6	32	114	0.7	(0.4-1.2)	11	62	0.4	(0.2-0.9)
7+	13	32	1.1	(0.6-2.4)	6	14	1.2	(0.4-3.5)

[a] Missing data are not included.
[b] Adjusted for age at death and socioeconomic status.

posity also directly affects estrogen metabolism to produce a more active form (estradiol), and it lowers the levels of sex-hormone-binding globulin (SHBG) to increase the amount of bioavailable estrogens.[30-32]

Epidemiologic studies of male breast cancer have suggested an excess risk associated with elevations in serum levels and urinary excretion of estradiol,[34-36] with men in the highest category of body weight having a 30 percent increase in circulating estrogens and a 39 percent decrease in SHBG.[36] In addition, a high proportion of male breast cancer patients have shown cytoplasmic estrogen receptors.[12] In addition to an array of risk factors suggesting the role of endogenous estrogens, it is noteworthy that elevated risks have been linked to exogenous estrogens through medicinal or occupational exposures.[37]

The NMFS questionnaire sought only limited information, due to anticipated recall problems with surrogate interviews. We had no data on certain known risk factors, including gynecomastia, Klinefelter syndrome, testicular diseases, or family history of breast cancer. However, it is unlikely that the association we observed with obesity can be explained by Klinefelter syndrome, which appears to account for only three to four percent of male breast cancer in previous surveys.[17] Since gynecomastia has been associated with obesity,[38] we could not clearly distinguish the role of obesity independent of gynecomastia.

Similar to female breast cancer, we found an excess risk of male breast cancer associated with increased SES, which appeared to be independent of obesity, diet, and exercise (OR for men in the highest SES adjusted for these factors is 1.7, CI = 1.1-2.9). In our study, SES showed a slight relation to BMI and exercise, but other unmeasured lifestyle factors correlated with SES may be involved.

In addition, our data suggested that consumption of red meat may increase the risk of male breast cancer, while intake of fruits and vegetables may lower risk. Although meat consumption may act in part by increasing body weight, further studies are needed into the dietary components and the nutritional/hormonal mechanisms involved in the etiology of male breast cancer.

We found no association with either cigarette smoking or alcohol drinking, although alcohol intake has been linked to female breast cancer.[39] It is worth noting that smoking and drinking usually are overrepresented in dead controls.[40,41] Although we excluded persons who died of alcohol- and smoking-related causes as potential controls, the prevalence of 'current' smokers among our controls (34 percent) was higher than that in the US population (25 percent) during the time period of this study.[42] This high frequency may have resulted in our underestimating the effects of smoking and drinking, so that further studies are needed.

Selection of controls has a direct impact on the validity of case-control comparisons and risk estimates, especially when dead or cancer controls are included.[43] In our study, 35 percent of the 512 controls died of cancer. However, very few controls died of endocrine-related cancers, since those with cancers of the thyroid or adrenal gland were not eligible to be included as controls. In addition, when controls whose primary cause of death was prostate cancer ($n = 62$) or diabetes ($n = 41$) were excluded from the analysis, results did not change materially. After these exclusions, some of the controls may have died from conditions related to endogenous hormones or obesity; the real association between obesity and male breast cancer is likely to be higher than that reported in our study, since inclusion of controls with obesity- or hormone-related causes of death would tend to underestimate risks associated with male breast cancer.

In summary, despite its limitations, our nationwide case-control study of male breast cancer pointed to obesity as a major risk factor, although there were also suggested links to SES, dietary factors, and exercise, as with female breast cancer. Further investigations are needed to clarify the interactions between nutritional, hormonal, and genetic factors in the origins of male breast cancer.

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